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Congestive Cardiomyopathy in the Canine

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Introduction

Diseases of the myocardium can be classified either as primary or secondary. Primary cardiomyopathies include idiopathic congestive or hypertrophic forms. Causes of secondary cardiomyopathies are primary, systemic diseases, the most common of which are infectious, metabolic, toxic, ischemic, and neoplastic diseases that cause some myocardial damage.

Idiopathic congestive cardiomyopathy of dogs was first recognized as a specific disease in 1970.¹ This disease is characterized by a progressive dilation of all cardiac chambers which results in decreased contractility and cardiac output. This leads to congestive heart failure and its compensatory mechanisms. As the atria continue to dilate, arrhythmias can be seen, the most common being fibrillation and premature ventricular contraction. These arrhythmias cause further reduction in cardiac output, thus increasing the degree of congestive heart failure.²

Congestive cardiomyopathy is also known to occur in other species as well as humans. Specific forms of the disease have been described in boxers and Doberman pinschers.^{2,3}

Incidence

Idiopathic congestive cardiomyopathy is the most common cardiomyopathy in the dog. Generally, the disease is seen most often in large and giant breed dogs. The most common breeds are listed as Great Dane, Doberman pinscher, Saint Bernard, Irish setter, English bulldog, Newfoundland, standard poodle, Great Pyrenees, Afghan hound, Scottish deerhound, bull-mastiff, Bouvier des Flandres,

greyhound, Labrador retriever, Chesapeake Bay retriever, and Gordon setter.¹

The disease may strike dogs between the ages of four months and 11 years, but it most commonly occurs in the middle years of three through eight.^{1,5,6} The incidence is much higher in males than females.

History

The historical complaints many times give a vague picture of illness. These dogs often present with anorexia, weight loss, weakness and general debility.⁴ The signs can be descriptive of right, left or combined heart failure. They would include abdominal distention caused by ascites, hepatomegaly, or splenomegaly, or even loose stools as a result of venous stasis in the gut.⁷ Owners may also describe respiratory signs such as coughing, hacking, and dyspnea.^{1,6} Other possible complaints are exercise intolerance and syncope.

The onset is usually subacute, including a gradual deterioration over a one to three week period. Also possible is a more acute type onset over a two or three day period. Most likely the sudden onset occurs after a prolonged period of subclinical disease, and leads finally to atrial fibrillation which forces a rapid decompensation and resulting signs of heart failure.²

Physical Exam

The physical exam often includes weakness, depression and general malaise. Respiratory signs will include dyspnea, orthopnea, and a moist, productive cough.^{2,8} Decreased capillary refill time and cyanotic mucous membranes are sometimes noted. The pulse is rapid (150–250 beats per minute) and irregular in rate and strength. During atrial fibrillation or ventricular premature contractions, there will be a pulse deficit. Ascites may be detected by abdominal distention and a fluid wave on bal-

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lottement.² Also seen are venous distention, engorgement of subcutaneous veins, and jugular pulses if tricuspid insufficiency has developed. On abdominal palpation hepatosplenomegaly is frequently present.

Auscultation

Over the lung fields coarse bubbling rales are heard, suggestive of pulmonary alveolar edema.¹ Often pleural fluid is recognized by muffled lung sounds below a fluid line.⁸

The heart sounds may be hard to evaluate due to the loud background respiratory noise and the rapid heart rate. An irregular tachycardia (up to 280 beats per minute) with detectable pulse deficit is often found. Dilatation of the heart may cause a moderate systolic murmur over the mitral valve area or a diastolic gallop.^{1,2} Characteristically with atrial fibrillation, the first heart sound intensity will vary from one beat to the next.⁸

Clinical Diagnostics

Blood serum parameters are usually not grossly abnormal, but severe congestive heart failure can cause some abnormal values. Prerenal azotemia can be caused by decreased renal perfusion. Hypoproteinemia and hyponatremia are the results of simple dilution of the fluid retained by the kidneys with decreased perfusion.⁸ Hepatic congestion may result in liver enzyme leakage. Elevated SGOT and LDH values are probably due to liver congestion rather than cardiac muscle disease.² Pleural and abdominal fluid are generally evaluated as true or modified transudates.²

Hypothyroidism has been reported as being a related condition in idiopathic congestive cardiomyopathy.^{2,8} Thyroid function tests may give interesting data, but the relative diagnostic significance, insofar as this disease is concerned, is as yet undetermined.²

Radiographically the major abnormality found is a moderately large to large cardiac shadow on chest films. Also, pulmonary venous distention two to three times normal size with rapidly diminishing size as the veins extend into the lung fields is a helpful diagnostic lesion.⁸ Left atrial enlargement can become big enough to displace the left main stem bronchus dorsally.⁸ On angiocardiography, researchers have found dye retention in the ventricles with no difference between end-systolic and end-diastolic volumes.⁹

Chest radiographs will often have evidence of pulmonary edema and pleural fluid. The ab-

normal findings are described as alveolar patterns or interstitial-alveolar patterns. If abdominal films are taken, hepatomegaly and the "ground glass" density of ascites are common findings.

The electrocardiograms of congestive cardiomyopathy dogs are generally helpful in diagnosis. By far the most common arrhythmia seen is atrial fibrillation. Atrial fibrillation is a supraventricular arrhythmia recognized by tachycardia (180–280 beats per minute), random R-R intervals, absence of P waves, and baseline fluctuations called F waves.^{2,8} The QRS complex can be normal or increased in amplitude and/or duration with left ventricular enlargement. Also, in the absence of atrial fibrillation, atrial enlargement (P mitrale or P pulmonale) can be seen.⁸ Generally, the mean electrical axis will be normal in these dogs.² In a smaller percentage of the cases (approximately 20%), and often late in the disease, ventricular premature contractions have been observed.^{3,8} S-T segment depression and T wave changes have also been observed.²

These findings may be the result of myocardial degeneration, or hypoxia and microinfarctions due to decreased coronary circulation.¹ Also described in rare cases is evidence of conduction disturbances.⁸

Differential Diagnosis

Other causes of right heart failure and ascites must be ruled out when diagnosing congestive cardiomyopathy. Dirofilariasis is an important differential diagnosed by microfilariaemia, right axis deviation on the ECG, and specific changes in pulmonary artery configuration on chest radiographs. Sometimes an occult heartworm test is needed to confirm dirofilariasis. Pericardial effusion or restrictive pericarditis should be considered with muffled heart sounds, cardiac friction rub, or elevated central venous pressure. Other rule-outs would include congenital cardiac anomalies as cause for heart failure or abdominal neoplasia and hypoproteinemia for ascites.¹

Therapy

Congestive cardiomyopathy has no specific cure, so therapy is aimed at clinical management for symptomatic improvement. Generally, the disease is managed as a congestive heart failure.⁷

There are four major goals of therapy to serve as guidelines: (1) attempt to strengthen

the heart muscle as a pump; (2) decrease the workload of the heart thereby sparing the pump; (3) prevent secondary damage to other organs as a result of heart failure; and (4) promote recovery of myocardial function.¹ Therapy will ultimately be a combination of treatments with each case being managed individually.⁸ In the acutely symptomatic patient, it is important to avoid stress to the dog, which might result in further decompensation. If necessary, acepromazine or morphine may be administered to reduce anxiety.

To strengthen the heart muscle as a pump, digoxin is used. Digoxin will reduce the heart rate making the pump more efficient. Also, the contractile strength of the heart is increased.⁷ The speed of digitalization is dependent on the severity of the condition. Rapid intravenous digitalization is reserved for life-threatening situations.⁹ It should be remembered that large breed dogs require smaller doses of digoxin per pound than used for other dogs.

Positive signs of effective digitalization are diuresis and reduced heart rate.^{8,10} The heart rate at full digitalization should be in the 80–120 beats per minute range. Also signs of toxicity, such as anorexia, vomiting, diarrhea, and cardiac arrhythmias, should be monitored.^{8,10}

If the heart rate is effectively altered with digoxin, a beta-adrenergic blocker such as propranolol can be used. Propranolol will help slow the heart rate, suppress ventricular premature contractions, and help restore normal sinus rhythm to atrial contractions.⁷ However, propranolol has a negative inotropic effect on the heart muscle, and thus should only be used with or following digoxin therapy.⁸

Decreasing the workload of the heart is an important objective of therapy, and this can be done in more than one way. Afterload can be decreased by vasodilators such as acepromazine. Preload can be reduced with diuretics.¹ Diuretics can also help reduce pulmonary edema and thus increase blood oxygenation.¹ Furosemide given intravenously is used in critical patients.⁹

In preventing secondary damage to other organs, perfusion and oxygenation are the keys. Digoxin and diuretics help considerably towards this goal; however, if the dog is cyanotic or severely dyspneic, oxygen therapy is indicated.⁹

To promote recovery of myocardial function, long-term management must be considered.

Dietary management with low salt or low sodium content feeds is advised to reduce the resulting fluid retention.¹⁰ These diets can be home-prepared or commercially obtained.¹⁰ Exercise restrictions also are necessary in long-term management to keep the cardiac work load down and to avoid catecholamine and sympathetic affects on a now-irritable myocardium.

Pathology

The most significant gross pathological findings are in the heart. Cardiac lesions include a large rounded heart, thin-walled, dilated chambers, ruptured chordae tendineae, endocardial jet lesions, dilated atrioventricular annular rings, and disseminated foci of myocardial necrosis.^{2,4,8,11}

The rest of the gross pathology found is generally related to a failing heart muscle. These findings include hepatic congestion, pulmonary congestion, ascites, hydrothorax, and infarcts in multiple organs.^{3,11}

On histological sections of cardiac muscle, the lesions found are subendocardial necrosis and scattered myocardial necrosis and fibrosis.¹¹ Also seen in cardiac muscle are irregularly-sized muscle fibers.³ Small to medium sized myocardial arteries have intimal and medial hyperplasia. Ultrastructural changes in the myocardial cells include sarcoplasmic vacuoles, lipofuscin granules, proliferated elements of sarcoplasmic reticulum and mitochondrial alterations.^{3,11} None of these ultrastructural changes are considered pathognomonic for congestive cardiomyopathy; however, many of these nonspecific changes of myocardial necrosis are seen in similar cardiac diseases in cats and humans.^{11,12}

Prognosis

Long-term survival of these dogs is unlikely, with most authors suggesting 6–12 months maximum survival time.^{1,7,8} It has been observed that the prognosis is generally worse in Doberman pinschers. Considering the grave prognosis, emphasis should be placed on correct diagnosis and therapeutic management as long as the dog can be kept comfortable. At that point in the progression of the disease, where the animal is judged to be suffering from decompensation unresponsive to treatment, the humane option may be euthanasia.

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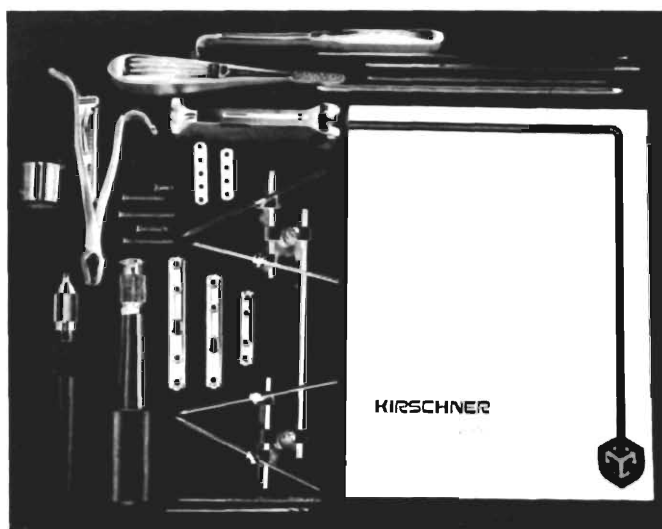
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